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"TWO THOUSAND OPERATIONS FOR APPENDICITIS,"

WITH DEDUCTIONS FROM HIS PERSONAL EXPERIENCE.

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It seems to me that a retrospective consideration of the subject of appendicitis might be of value to the general practitioner and the surgical specialist, as well as to the occasional operator in this disease. It would be useless to tabulate such a large number of cases unless all of the phenomena of etiology, symptomatic manifestations, clinical course, technical procedure, pathological conditions, and final results were included. This would mean an enormous labor from which, I believe, there would be no adequate return. The first operation, in its modern sense, was performed by me March 2, 1889. The two-thousandth was completed July 22, 1903.

I will refrain from going into the history of the struggles of the pioneers in the operative treatment of appendicitis. There is no procedure in surgery where the battle was so fierce and so continuous and where the statements of the uninformed were so personal, so galling, and so unjust as in the contest for and against early operation in acute infective appendicitis. Looking backward one can scarcely comprehend how a so-called intelligent profession was so slow in accepting the overwhelming force of numbers and facts, which could not be altered by theory or speculation. It is, however, but a repetition of the past and an index of the future; the medical profession, as a body, always has preferred to theorize and dream rather than analyze, investigate, or accept facts. This is particularly true of the pathological conditions of internal organs, and what could be said regarding the attitude of the profession

toward the diseases of the appendix in March, 1889, can be said to-day of diseases of the stomach, the pancreas, and, with little reserve, of the kidneys.

The diversity of opinion expressed as to the presence or absence of pus in acute, infective appendicitis, in the discussions in 1889, 1890, and 1891, is most interesting reading for the student of medical progress. Practitioners whose cases recovered from the attack insisted that they were catarrhal inflammatory processes and recovered without the formation of pus, notwithstanding the evidence produced by every operator of that time that pus was present in all of the acute infective cases operated in the early stages—now a generally recognized fact. Every one recalls how reluctantly the advocate of the soothing, death lullaby of the opium treatment vacated his position, and how equally persistent and belligerent was the advocate of the death-groaning calomel and castor oil participant; the still unconquered, nine-lived procrastinator has not yet capitulated, each and every one of these standing out against the most convincing presentation of pathological phenomena.

Surgical Anatomy. The appendix, from a surgical standpoint, can be considered as located almost universally in the right lower quadrant of the abdomen, between a line drawn from the umbilicus to the symphysis and one drawn from the umbilicus to the tip of the eleventh rib on the right. The base of the appendix is usually found a little above a line drawn from the umbilicus to the anterior superior spinous process of the ilium and about two-fifths of the distance from the process (McBurney's point). In inflammatory conditions the appendix is usually found extending downward from this point. In children it is usually higher up. It may even approximate the costal arch and become attached to the margin of the liver or gall-bladder, or rest behind the caput coli. Once I found it between the liver and the abdominal wall. Below, it may be closely adherent to the inguinal tract, hanging over the margin of the pelvis; or, indeed, be found in the vesicorectal or Douglas' pouches. When the point of extreme sensitiveness cannot be determined in the usual position, examination of the loin and rectal and vaginal examinations should never be overlooked, as in about 5 per cent. of the cases I have found the appendix in the pelvis, and in a large proportion of these the abscess was entirely confined to the pelvis, not always to the right half of the pelvis, but filling in the entire pelvis, the vesical tenesmus and urinary retention indicating its location. In only two cases did I find the appendix to the left of the middle line. Once it was adherent to the left tube and the second time to the transverse colon. It was rarely found behind the peritoneum, although abscesses in the retroperitoneal space, from acute appendicitis, were not rare, due to perforation and not to malposition of the appendix. It is many times embedded in the wall of the caput coli. When found in that position in the

intermediate operations I have always considered it, not an anatomical anomaly, but a pathological sequence. In the intermediate operations, as well as in the acute stage, it is occasionally found attached to the ileum. Twice I found it strangulated and gangrenous in femoral hernia, and a number of times, in the acute and intermediate operations, in inguinal hernial sacs. The presence of acute infective appendicitis should be suspected in all right-sided hernias, where there is rapid elevation of temperature, with nausea and vomiting, shortly after the manifestation of strangulation, as simple intestinal strangulation in a hernial sac never gives a primary elevation of temperature. I have never found the appendix in an umbilical hernia.

The length of the appendix varied greatly—from three-quarters of an inch to six and three-quarter inches. The mesoappendix is from two and a half to four inches in length. In pathological conditions it may be entirely absent; that is, the appendix may be adherent to the intestine or to the abdominal wall. The mesoappendix is attached for about two-thirds of the length of the appendix; occasionally it is of enormous thickness, containing large deposits of fat. Many times the tip is adherent and an opening exists between the tip and the mesentery. I have had four cases of intestinal obstruction through this opening.

The proximal end at the valve of Gerlach is usually the smallest portion of the lumen. In acute infections of the mucosa, this opening becomes occluded from the œdema, producing retention of the infective material, as manifested by the acute primary colic and reflex primary nausea. The most severe infection and necrosis of the mucosa, however, does not occur at its most narrow part, but most frequently at about two-fifths of its length from the tip toward the base. The lumen of the body of the appendix varies greatly. Where it has been subject to high retention with subacute infection it enlarges gradually; where it has been subject to accumulating pressure with occlusion at the base without infection, it enlarges greatly to the size of the adult thumb or even larger. When the infection is severe the infiltration is great, and in these cases the subsequent cicatricial contraction diminishes the size of the lumen below its normal anatomical capacity, even to complete obliteration, when the epithelium is destroyed. These are the same pathological changes that take place in all of the hollow viscera under the same conditions, and particularly in the gall and urinary bladders.

The appendix has no vessel running parallel to its long axis; the vessels of the mesoappendix run directly through the mesentery to the appendical wall. At the mesenteric attachment there is no peritoneum. This is the weakest point in resistance under infection, and is the most common location of perforations where foreign bodies are present.

ETIOLOGY. When the disease was first recognized as a surgical affection, the acute infective lesions were considered to be due to the usual causes which produced enteritis in the remaining portion of the intestine and it was considered essentially a disease of the summer months. Now since we have become more familiar with it we find that it is almost as common in winter as in the summer. We find that it does not come on under the same climatic or dietary conditions as the other types of enteritis; that it most commonly follows exposures such as would produce so-called "cold" of the respiratory tract. So commonly is it a sequence of such exposure that a very prominent Chicago physician at one time considered it essentially a rheumatic manifestation. This, however, we do not accept.

Foreign bodies, as fruit seeds, gallstones, capsules, etc., were present in a little less than 2 per cent. of the cases. Fecal concretions were found in 38 per cent., and we believe that the erosion of the appendical mucosa by these foreign bodies produces an atrium for the admission of the infective flora into the tissue and precipitates under favorable conditions the acute attack. Indiscretions in diet appear to have little if any effect as an etiological factor.

The types of infective flora found in cultures and stainings from the appendices vary in the following order of frequency: bacillus coli communis, staphylococcus pyogenes aureus and albus, streptococcus, pneumococcus, bacillus tuberculosis, actinomycosis.

Appendicitis occurs in all classes with about equal frequency. In my work it has been a little more frequent in males than in females. I do not consider that it is contagious or infectious. It does seem, however, to have family predilections. This I would rather consider due to the conformation of the appendix or the diminished resistance to infection in some families and the comparative immunity to infection in others. I have not infrequently had two members of the same family in bed with the disease at the same time.

SYMPTOMATOLOGY. The symptoms in the order of their occurrence may be mentioned as: first, pain in the abdomen, sudden and severe, followed by (second) nausea or vomiting, even within a few hours, most commonly between three and four hours after the onset of pain; third, general abdominal sensitiveness most marked in the right side, or more particularly over the appendix; fourth, elevation of temperature, beginning from two to twenty-four hours after the onset of pain. In severe cases the temperature reaches 102° to 103° between eight and twelve hours after the initial pain. The symptoms occur almost without exception in the above order, and *when that order varies I always question the diagnosis.* If the nausea and vomiting or temperature precede the pain I feel certain that the case is not one of appendicitis. Where the

temperature alone precedes the pain for a day or even two or three, then I am always fearful that it is typhoid fever with a typhoid ulcer in the appendix. I have had seven cases of this class; the first three were operated and typhoid ulcers were found in the appendix; the last four were not operated; all went through classic typhoid courses after the appendical manifestations and all seven cases recovered. Where sensitiveness has been present for a number of days, and particularly where it is low in the pelvis, I have found the focus other than appendical—that is, due to infection of the tube, tuberculous peritonitis, etc.

Pain was a constant and uniform symptom and usually colicky in character, although patients varied in their expressions of its severity. In not a single case was it absent as an initial symptom. It usually reached its acme of intensity about four hours after its onset. It subsided gradually in the majority of cases. When it subsided suddenly, as it sometimes did, within the first thirty-six hours, the subsidence was due either to liberation of the infective material through the neck of the appendix into the caput coli, rupture of the appendical wall, or complete gangrene of the appendix. Under these conditions the pus pressure was relieved and the pus absorption diminished or stopped. With the cessation of pain in these cases, whether it was due to gangrene, intestinal escape or perforation, came the diminution or cessation of absorption, *but not* necessarily a *diminution* or *cessation of danger*, as is so commonly though erroneously interpreted, and often with fatal result.

The secondary pain, after the first thirty-six hours, is usually not colicky in character, but of the typical inflammatory type and due to periappendical involvements. Severe pain after the primary subsidence is always a signal of great danger, as it announces a beginning peritonitis from perforation.

The primary nausea and vomiting is reflex, due to overdistention of the appendix from the accumulating products of the infection, and is the same type of nausea and vomiting that we have with stone suddenly impacted in the neck of the bladder or suddenly passed into the ureter. There is usually only one or two efforts at emesis and the nausea then passes away. The secondary nausea and often persistent vomiting is due to the periappendical involvement; that is, infection of the peritoneum, and is of the same character and often the same persistence as vomiting from perforation of the tube, intestine, or stomach, into the peritoneum. In extensive peritoneal infections the vomiting continues to the fatal termination, increasing in frequency as time advances and as absorption increases.

The abdominal sensitiveness is first diffuse, although the abdominal wall is not particularly rigid. When the appendix becomes fully distended and tense it will not tolerate pressure and is pro-

tected by a marked rigidity of the abdominal muscles, the same as the gall-bladder and urinary bladder are protected under similar conditions when overdistended. As soon as the acute tension subsides, the general sensitiveness disappears. It becomes circumscribed to the area of the appendix, whether it be at McBurney's point, in the subhepatic space, or in the pelvis. When absent above, the pelvic examination should never be overlooked. If the sensitiveness has been once circumscribed to a small area and increases suddenly, preceded by pain and by secondary nausea and vomiting, it strongly indicates a perforation of the circumscribed adhesions and an infection of the general peritoneum. In endeavoring to palpate the appendix, after the first twenty-four hours, the greatest care and caution should be exercised. Severe pressure is likely to produce separation of friable adhesions that are of life-saving importance.

Temperature was not absent in a single acute infective case in its *early stage*, that is, in the first thirty-six hours after the onset of the symptoms. In the acute severe infections it is present in a few hours. In acute obstructions of the neck of the appendix, with mild infection, it appears later. In the calcular obstructions it does not appear until about the twentieth to the twenty-fourth hour after the beginning of pain. It is always present in the early stage of acute appendicitis. I would not operate on a case where I was confident that no temperature had been present in the first thirty-six hours of the disease. I came very close to making an unnecessary laparotomy in a case of this character. The patient had typical acute onset of pain, nausea, and vomiting, local sensitiveness and tumor; so sensitive was the tumor that manipulation was impossible. She was a doctor's daughter. Her father insisted that there had been no temperature at any time, but I felt that it was a case of the proverbial shoemaker's child, and that attention had not been close enough to warrant the statement that no temperature had been present. After she was on the table and I had taken up the scalpel, I laid it down again, saying that I had never had a case without elevation of temperature. With some manipulation the tumor disappeared under the costal arch and proved to be an acutely displaced kidney; no operation. When the patient recovered from the anæsthetic she informed us that in play at a picnic the second day previous she was rolled across the edge of a board fence, the pressure being from behind forward, just below the last rib. It caused her severe pain in her side and the pain continued from that time. This produced an acute displacement of the kidney with the symptoms of pain, reflex nausea, vomiting and extreme local sensitiveness, but with absence of temperature.

The temperature may all subside within twenty-four hours after the onset, and if it subsides suddenly I am fearful that the cause of its subsidence is a gangrene of the mucosa, with failure of further

absorption and therefore an intermission of temperature. Where it subsides gradually from thirty-six to fifty-two hours after the onset, then I believe it is due to a cessation of pressure from rupture of the appendix either into the mesoappendix or to circumscribed adhesions around the appendix. The temperature here, as in other places, must be recognized not as a manifestation of pus, but as a manifestation of absorption of the products of infection; without the absorption there is no elevation.

A remission of temperature of one or two or three degrees with subsequent gradual elevation means an infection of a new area of tissue. The temperature may go below 99° and remain there after the initial elevation, and still a large quantity of pus be present, either circumscribed or diffuse, in the peritoneum.

Secondary elevation of temperature should always be noted with apprehension, as it indicates a fresh invasion of tissue, a thrombophlebitis, a peritonitis, or cellular infiltration. These elevations are indications for immediate operation, and that usually means immediate drainage. Under these circumstances no extensive laceration of tissue or separation of adhesions should be attempted for the purpose of making a complete operation. The focus of pus should be drained and, as a rule, nothing more done

Leukocytosis. For a time it was believed that much valuable information could be obtained by ascertaining the excess of leukocytes. The more extended our experience, the less valuable it became. Leukocytosis may be considered a blood reaction to the absorption of septic products. Infection may be present without producing the blood reaction. A sudden and great increase in the number of leukocytes, supported by the other symptoms, is usually indicative of an extensive peritonitis.

Pulse. The character of the pulse has little value in the differential diagnosis of appendicitis. In children, when there is rapid absorption the pulse rate is very high. In the later stages of peritonitis, when it becomes compressible, rapid, and feeble, it is a fair index of the degree of intoxication and a guide to the prognosis rather than to the extent of the inflamed area.

In two cases of perforative appendicitis, where the perforation had occurred a number of hours before, the patients were walking around, with normal pulse and normal temperature. A considerable quantity of intestinal contents was free in the peritoneal cavity. One of these patients walked into the hospital with his doctor, did not appear materially sick, though the perforation had taken place eight hours before he presented himself. There had been some nausea and pain immediately after the perforation. That subsided and there was no change in his pulse or temperature. He was put on the table, the abdomen opened, and a large quantity of serepurulent material was found in the peritoneal cavity; also a con-

siderable quantity of fecal matter, which had leaked through a calcular perforation in the appendix.

Tumor. In the very early hours of appendicitis, while the inflammatory process is still confined within the appendix, an examination of the abdomen reveals an apparent tumor. The centre appears to be hard and fixed. It is oblong and about the size of the thumb. One feels convinced that the appendix rests close to the abdominal wall. The impression of tumor under these conditions is due to the spasmodic contraction of the muscle fibres directly over the situation of the appendix. When the patient is placed under the anæsthetic the tumor entirely disappears and, indeed, the appendix may be in the retrocæcal position and many inches from the abdominal wall. So frequent is this deception that I feel it should be constantly borne in mind. In the subacute cases the enlarged, swollen, and distended appendix can be frequently outlined by careful examination. As the periappendical tissues become involved and the quantity of infective material increases, the tumor becomes more pronounced and often attains considerable size. This is usually located in the right iliac fossa, but its absence from that position, with a clinical history indicating an appendicitis, must not be construed as meaning that the tumor is not present, because it will be found to be in the loin or low in the pelvis.

Fluctuation is only present when there is a large circumscribed abscess in the later stages of the disease. In the early stages of the disease the absence of peristalsis in the appendical region is of value in indicating the nature of the process. A careful examination with the stethoscope reveals a "still" area for many inches around the appendix.

The size of the tumor bears little relation to the quantity of pus. The area of infiltration around a streptococcus or staphylococcus infection is small, while the area of infiltration around the colon bacillus group of infections is always very large in proportion to the quantity of pus.

It is needless for me to go into details concerning the other minor corroborative signs of appendicitis, or to go into the many complications in the symptomatology of the later stages of the disease. The greatest difficulty is experienced in making a differential diagnosis between appendicitis and acute catarrhal conditions of the caput coli, or acute retention conditions in the caput coli, especially associated with the intestinal type of la grippe. Here we have the pain, nausea, and vomiting, the local sensitiveness and the elevation of temperature all combined, and so closely does it resemble appendicitis that in two cases I have erred in the diagnosis. In both there were evidences of previous attacks of appendicitis, with adhesions of the appendix, but in neither were there manifestations of an acute process in the appendix. Nor have my errors taught me a means of preventing their recurrence. I believe I

have avoided many errors in diagnosis by the stress which I placed on the order of symptoms as they occur, namely, pain, nausea and vomiting, local sensitiveness, and elevation of temperature; a deviation from this order causes me to seriously doubt the diagnosis of appendicitis.

Clinical Course. The clinical course of acute infective appendicitis is fairly uniform. In the acute infectious cases without calculus the mucosa becomes infiltrated and œdematous within an hour or two after inoculation. The œdema and infiltration increase rapidly and the compression of the inelastic coats of the appendix with the assistance of the biotic and toxic effects of the pathogenic flora produce a rapid gangrene of the mucosa, submucosa, and occasionally the entire thickness of the wall, with now and then complete gangrene of the appendix, so that a perforation of the appendical wall, on the average, takes place in from thirty-two to forty hours in virulent infections. Infection with the presence of a foreign body (fecal concretion) is usually associated with only a small area of gangrene, and that at the point of pressure of the calculus; here there is a local necrosis or gangrene with perforation and leakage; perforation in these cases takes place a little earlier than in the infections without a foreign body. Occasionally pressure ulceration and perforation of the appendix takes place at the seat of a coprolith without an acute infection. In this last class of cases the first announcement of the trouble is made by the initial symptoms of the perforation. These cases represent about 1 per cent. of all of the acute cases. It is a dangerous type for the patient, as the initial symptoms are attributed to a beginning appendicitis, when in reality they are due to a beginning perforative peritonitis.

In the streptococcus infections the mesoappendix and neighboring tissues are more seriously involved and the area of gangrene is less than with the colon bacillus or the staphylococcus types. The pain is most severe in the first six to fourteen hours; it becomes less as the necrosis advances; the temperature is highest from the sixteenth to the thirtieth hour, preceding the completion of the gangrenous process. If the gangrene continues to advance, the temperature remains up; if it becomes stationary, the temperature declines.

In one case of complete gangrene of the appendix the temperature was 104° nineteen hours after the onset of pain; it was 99.2° twenty-six hours after, at the time of operation. The appendix was gangrenous throughout its entire wall to its junction with the cæcum. It was a black, shining mass without an adhesion and slipped into the abdominal incision. The necrosis was so rapid and complete in this case that there was no effort on the wall of the appendix to fix itself to the neighboring tissues. I regard with alarm a sudden drop in the temperature and interpret it as meaning

a cessation of absorption, due, most commonly, to perforation of the wall of the appendix, with diminished pus pressure; second in frequency, to the escape of the contents of the appendix into the intestine; third, to gangrene of the wall; fourth, to circumscribing tissue infiltration with diminished absorption (local immunity); and, fifth, diminution of the virulence of the flora in accordance with the law of putrefaction and fermentation.

After fifty hours the clinical picture changes from that due to lesions of the appendix to that of infections of the periappendical regions. If the perforation takes place into the mesoappendix, or under circumscribing adhesions, the clinical presentations are those of a limited infection, while if the perforation takes place into the free peritoneal cavity the symptoms of a more or less severe peritonitis manifest themselves, depending upon the types of infection and the degree of involvement.

After a circumscribed abscess has formed, the temperature, pulse rate, and pain subside to a considerable degree; indeed, the temperature may approximate normal as the infiltration in the abscess wall becomes more and more pronounced and absorption proportionately diminished. If the rupture be into the postperitoneal cellular tissue, then there is rapid rise of temperature from the absorption. If the rupture be into the free peritoneal cavity, the rule is, first, a primary drop of temperature as the pus is relieved of pressure and the peritoneum has not yet been abraded of its endothelium, and therefore does not allow rapid absorption. In a staphylococcus infection of the milder type the peritoneum is first covered with an exudate, which may go on increasing in thickness to the formation of a membrane resembling the diphtheritic exudate. This exudate lessens the immediate absorption and protects the patient against an overdose of the septic products. It paralyzes peristalsis and produces coprostasis, but in a short time this plaque loosens, is thrown off, and carries with it the endothelial covering of the peritoneum, leaving a distinctly abraded or blistered surface from which there is rapid absorption and a sudden collapse of the patient, frequent bowel discharges, anxious expression, and shortly death. This sudden change in the staphylococcus infection of the peritoneum takes place between the evening of the fourth and the morning of the sixth day. If the primary infection of the appendix be of the streptococcus type and it ruptures into the peritoneum there is rapid blistering of the peritoneum and, within a few hours, symptoms of intense intoxication, high pulse, anxious expression, talkative delirium, quick perceptions, tympany, and all the manifestations of severe and acute streptococcus intoxication.

The action of the colon bacillus, when it gains entrance into the peritoneum, may be said to be midway between these two pictures. At times it produces but slight irritation, mild elevation

of temperature, considerable seropurulent exudate, and a gradually increasing disturbance of the surface of the endothelial covering of the peritoneum. The advancement is so slow that the tendency with this milder type is to circumscription of the process by peritoneal adhesions. On the other hand, under certain conditions, which we little understand, the colon bacillus may have a powerfully irritating effect on the peritoneum, produce rapid destruction of its lining cells, and expose its lymphatic stomata for rapid absorption. Indeed, its virulence may be so great as to produce gangrene of the peritoneum and even of the entire intestinal wall. This is fortunately rare, but is an extremely fatal type of the action of this bacillus. Sometimes the gas bacillus is associated with it and the tissue becomes emphysematous. There is not usually a high temperature in this class of cases, but great physical depression, lowered arterial tension, sunken eyes, somnolence, and finally low muttering delirium, differing entirely from the clinical picture of the streptococcus intoxication.

The quantity of pus found in the peritoneal cavity in the dry staphylococcus type is usually small. It may be large in quantity, and then it is of a seropurulent character. With the streptococcus type there is little, if any, free pus, but the peritoneum has that peculiar dry, granulating, blistered appearance which was so frequently seen in the post-partum sepsis of the pre-antiseptic period. The colon bacillus produces pus with an offensive odor, usually considerable in quantity and thick and creamy in character. The intestines are agglutinated together and many times separate pockets are formed. The elevation of temperature, the tympany, and the collapse are commonly slower in appearing with the colon bacillus than with either of the other types of infection. Collapse is not a sign of perforation, but a manifestation of the absorption of products of infection by an abraded or blistered peritoneum. It will appear rapidly or slowly after perforation, depending on the virulence of the infection and the rapidity with which the endothelium of the peritoneum is destroyed. This has been one of the most difficult lessons for the general profession to learn. For years we believed that collapse was an immediate manifestation of perforation; we must now recognize that it is a symptom of septic intoxication and always a late symptom, as far as the clinical course is concerned.

It can be seen from the various pathological conditions mentioned in the clinical course that a case of appendicitis may subside within a few hours by escape of the infective material along the tract of the appendix into the cæcum; that it may subside as a circumscribed inflammation of the appendix; that it may form a circumscribed infection without the production of pus in the free peritoneal cavity, with an inflammation only of the lymphatics of the mesoappendix and local peritoneum; that it may subside as a circumscribed

abscess outside the appendix, rupture through the wall of an adjacent coil of intestine, and empty itself in that way; that it may subside as a circumscribed peritoneal abscess and remain quiescent to be opened later; that it may terminate in a circumscribed or diffuse general suppurative peritonitis; that while it is a circumscribed abscess there may be a thrombophlebitis of the iliac veins, and the general venous circulation become infected with the manifestations of embolism, infarcts, and secondary abscess; that a portal venous branch may thrombose and hepatic abscess or pylephlebitis result; that there may be continuations of the infective process by contiguity of tissue or continuity of surface, extending down into the pelvis and up the opposite side of the abdomen, upward behind the peritoneum and around the kidney, upward beneath the surface of the liver and around the gall-bladder, or into the subphrenic space, through the diaphragm into the lung, and the pus be expelled through the mouth; or it may rupture into some portion of the urinary or genital tracts. All of these later and grave conditions are the sequences or the direct result of *omission* on the part of the surgical or medical attendants who had charge of the case during the first forty-eight hours of the appendicitis.

The closest attention has been paid in all of these cases to the relation between the symptoms and clinical course and the pathological findings, so that we may better anticipate the intra-abdominal changes by the symptoms and physical signs, or at least that we may know when and under what conditions pathological changes are not announced by appreciable symptoms, and thus avoid fatalities and surprises by a proper interpretation of the presence, order, or absence of symptoms.

I will not mention here the clinical course produced by the great variety of displacements, flexions, retentions, adhesions, torsions, and abnormal fixations of the appendix, as that would carry me beyond the scope of the paper. I must state, however, that we are just beginning to appreciate the great disturbance that can be produced in the intestinal tract and in the general metabolism, and the distressing and complex train of symptoms which result from these minor appendical lesions. We must operate much more frequently in the future for their relief than we have in the past, as we are learning from clinical experience that the disturbances produced by them are out of proportion to the logical deductions that could be made from the pathological conditions. The completeness of cures by operations on this class of cases is one of the most gratifying in the entire field of appendical surgery.

TIME FOR OPERATIVE INTERVENTION. The time for operative intervention may be divided into four stages: first, early, within the first forty-eight hours; second, in the active increasing inflammatory process, from the second to the fifth day; third, in the sub-

siding inflammatory process, from the fifth or seventh day on; and, fourth, in the intermediate stage—between attacks. From the previous statements concerning the clinical course and pathological changes, it can be seen that the most favorable time for operation is within the first forty-eight hours of the attack; or, from a pathological basis, before the perforation of the appendix or the infection of the periappendical tissue. The diagnosis can and should be made with accuracy in the great majority of cases before the end of the first twenty-four hours, and almost universally within the first forty-eight hours. From the symptoms and clinical course of the disease in the first forty-eight hours it is impossible to predict, with any degree of certainty, what the subsequent course of the case will be—that is, whether the tendency will be to subsidence and cure by natural processes or to a virulent, if not fatal, termination. This is the consensus of opinion of the large majority of those surgeons of greatest experience.

The danger of operative intervention in the early stage is scarcely more than that of an exploratory laparotomy. The time required for the convalescence is not more than two and a half to three weeks. Drainage is, as a rule, not indicated and hernia improbable. Unnecessary operations as a result of error in diagnosis would be very limited. The patient would, therefore, be relieved of his appendicitis without hazard, without prolonged illness, without the danger of unpleasant sequelæ, without the possibility of recurrence, by the only timely operation. To me there appears to be no excuse, no explanation, no logical process, no justifying hope that relieves the patient of the dangers of this disease. Procrastination, under these circumstances, I do not interpret as a manifestation of knowledge, experience, judgment, or true conservatism, but a stigma of their opposites. This should, therefore, be considered the period of election.

Should we operate in the second stage, during the increasing or spreading inflammatory process, which may mean anywhere from the second to the fifth day? In this stage we may have the circumscribed abscess around the appendix, the active inflammatory process of the neighboring tissues or organs, or the early pathological changes of a circumscribed or general peritonitis. We often find the temperature and pulse high, meteorismus, intestinal paralysis, and acute infected tissues, with manifestations of severe intoxication. These patients will not stand extensive manipulations. They are already fully burdened with the toxins, and this intoxication must not be increased or the patient will be sacrificed. Shall we operate? Yes, and in this stage. But the operation must be a limited one; that is, simple opening and relief of pus tension in the infected area with the removal of the appendix, if it be accessible and easily amputated. There should be the least possible separation of agglutinations or other trauma to the infected tissues. Agglu-

tinations and adhesions are life-saving, both in circumscribing the process and in rendering the local tissues unfit for absorption. Many of the fatalities in operations for appendicitis have been due to the failure of recognition of these facts. Where the patient is apparently overwhelmed with the intoxication from a circumscribed or diffuse peritonitis or inflammatory process, I content myself with making a simple incision in the abdomen and relieving the pus tension by the insertion of a large drainage tube without irrigation, without sponging, and without manipulation of the tissues. On the other hand, in the ascending stage of the disease, where the depression is not noticeable, when the intoxication is not severe, even when the quantity of pus is large, circumscribed or not circumscribed, the appendix is removed. In other words, the extent of operation is governed rather by the constitutional symptoms of the sepsis than by the extent or character of the pathological changes. One fact must never be lost sight of in operating on this class of cases, and that is, that the pus is usually virulent, and when organized adhesions are present they must not be separated, as they expose the surface to acute absorption. Organic adhesions are rarely formed before the fifth day. Exudates must not be rubbed nor torn off, as they carry with them the endothelia and leave an abraded absorbing surface. The peritoneum should be considered similar to the skin; in the latter, while the epithelia are intact, there is practically no absorption; they are the shingles of protection; with the peritoneum, while the endothelial cells are intact, there is practically no absorption. The rule, therefore, which I follow is not to treat any of the active inflammatory conditions on the expectant plan, but to relieve the suppurating infected centres by simply opening and inserting an efficient drain, and in the cases where the constitutional reaction is mild and the appendix accessible, it is removed. In my work the appendix has been removed in nine cases out of ten operated in the second stage, while if attempted in the 10 per cent. of selected cases, with severe symptoms of constitutional sepsis, the majority of them would be fatal. In the last two and a half years' work in general septic peritonitis I have had but one death following the operation, and that was a case of appendical origin, operated on the fifth day. The patient succumbed six days after the operation with a double pneumonia. The peritonitis had entirely subsided. This was the only death that occurred in any of the cases of peritonitis operated, including the typhoid, gastric, and intestinal perforations and sixteen consecutive cases of general suppurative peritonitis. The treatment after the operation in these cases consisted in placing the patients in a semisitting position, 35 to 45 degrees, retaining them in this position for three or four days, administering large quantities of saline solution (per rectum), from four to twelve quarts in twenty-four hours; the saline must be allowed to seep in, the tube remaining

constantly in position. No water was given by the mouth. The stomach never absorbs water, and under these conditions it does not readily transmit it to the intestines for absorption. Mild catharsis was induced with small doses of calomel, beginning eight hours after operation.

I am convinced that the great mortality which has been reported in operations in the second stage and under the clinical conditions mentioned above have, in a measure, been due to excessive manipulation, sponging, flushing, adhesion separating, and *prolonged operation*. The deaths in these cases of acute infection are due to the depression resulting from a sudden absorption of an overwhelming dose of toxin; this absorption is favored by the manipulations mentioned.

Let us contrast the treatment of an acute infective focus in the peritoneum with the treatment of a similar infection; for example, osteomyelitis in the ends of long bones. In the latter we open or tap without irrigation, sponging, curetting, or manipulation, and simply insert a drain. If we will treat the peritoneum with the same consideration we will have equally gratifying results. Water has no more license in the peritoneum than it has in the lung; the same may be said of trauma.

The posture after operation allows the pus to settle in the most dependent portion of the peritoneum; there it is pumped out through the tube by the alternating respiratory pressure. During transportation of cases of general suppurative peritonitis to the hospital the patients are kept in a sitting position; during operation they are kept in the same position. I do not consider any of these steps in the sense of life-saving stitches, but their combination and the results obtained have revolutionized my ideas and prognoses in general suppurative peritonitis, as well as in the acute and viciously septic appendicitis. Nor would I expect to cure a case of peritonitis in the stage of projectile, black vomitus; cold, clammy skin; pinched expression, ballooned abdomen, and dusky, cyanotic hue. Here, the fatal dose is already in the circulation, and inhibition, antitoxic eliminants, and hematodiluents would have no effect.

Operations in the third stage, or the stage of subsidence of the inflammatory process or retardation of the process of absorption, are not at all as urgent as in the first or second stage. To begin with, the destructive process has been overpowered by the local resistance of the tissue (the local immunity of tissue infiltration), and an effectual encapsulation has taken place; or they have been emptied through ulceration into a neighboring coil of intestine; or they are slowly destroying their boundaries in the line of least resistance and little absorption is taking place.

This process may continue for a considerable period of time unless some accident should rupture the wall, or the pus should come in contact with some vein and thrombophlebitis with embolism

ensue. The retained products of infection under these circumstances are always an element of danger, and in the hands of judicious and careful operators in this stage they can be conducted to the surface without hazard to the patient, and the appendix can, very frequently, be removed without extensive separation of adhesions or scattering the pus over non-infiltrated areas by carefully coffer-damming the operative field. If a mass is felt in the iliac fossa, pelvis, or loin, and is readily accessible in any of the positions, it should be opened directly over the most prominent point and drained. This, however, must always be the exceptional route. When a case has progressed to this stage, the patient is in a position to be placed in the best operative environment and to have the best skill. Personally, I open through the anterior abdominal wall in practically all of these cases and coffer-dam the field before opening the pus cavity. I have had six deaths in this class of cases, two in which the peritoneal cavity was not opened, but where the bulging abscess was simply opened and drained. In both of these the opening was made in the anterior abdominal wall without entering the free peritoneal cavity. Either from the preparatory manipulation or from pressure on the abscesses during operation they ruptured below into the free peritoneal cavity. Both were large abscesses, one in a fourteen-day and the other in an eleven-day case. The accident was not detected, as the pus flowed freely from the abscesses when they were incised. They died seventeen and twenty-six hours respectively after operation, from acute septic peritonitis. These cases occurred years ago, and since that time I never open directly into an abscess through an anterior incision. I always keep to the central side of the abscess and open into the free peritoneum, placing the coffer-dams before liberating the pus.

Three were operations in the third stage, with a secondary elevation of temperature and manifestations of absorption; two of these died from acute sapræmia from excessive separation of organized adhesions in efforts to remove the appendix in the presence of virulent pus. The other died from advancing gangrene with aërobic infection in which the intestines and local tissues sloughed. The sixth died of pylephlebitis with multiple hepatic abscesses. This I do not feel could be attributed to the operation, but to the fact that the operation was late and a thrombus had formed in the portal vein.

In the third stage the presence of pus is always an element of danger, and we should not feel content until it is removed.

In the fourth or intermediate stage the question arises, Should we operate on every patient who has been through an acute attack of appendicitis? If so, why? The reasons for operating that may be given are these: first, after an attack of appendicitis the patient is prone to recurrence; second, upward of 60 per cent. of the cases operated by me in the intermediate stage had had more than one

attack; third, in the large proportion of the cases in which there was only incision and drainage of the abscess (which was a procedure frequently recorded by me in my first three hundred cases) the attacks recurred. In one patient I drained a periappendical abscess on three different occasions, always with severe sepsis, before I succeeded in inducing the patient to come in between attacks to have the appendix removed; fourth, the pathological changes, such as adhesions, flexions, stenoses, etc., following a primary attack, very much favor a recurrence; fifth, even if there was no danger of recurrence of the inflammatory conditions, the adhesions, fixations, stenoses, flexions, and retentions in the appendix produce sufficient disturbance of the digestive tract to demand an operation for their prevention; sixth, recurrent infections are, practically, as dangerous as primary attacks (I recall one patient who died in the seventh attack); seventh, the appendix can be removed in the intermediate stage without danger. About two-thirds of the entire number of operations were in the so-called intermediate stage. In all of these I had one fatality and that was from an acute, infective peritonitis, traceable to an interne who had, without my knowledge, made a post-mortem on a post-abortion peritonitis the night before the operation; two other operations performed on the same day were similarly infected. I had one case of severe, but not fatal, hemorrhage, in the person of a physician, which was apparently due to penetration of the deep epigastric artery by the closing suture. This would mean about one death in 1300 or 1400 operations. In four of the intermediate cases only a stump of the appendix remained, as it was destroyed by the sloughing process. This is an extremely small number when one considers the severity of the inflammatory process and the number of cases in which the mucosa is gangrenous, as seen in the early operations. A partial or even complete obliteration of the lumen of the appendix is not rare.

THE TYPE OF OPERATION, as far as its application to many of the pathological conditions, has been mentioned from time to time in the progress of the paper. The routine incision is through the outer margin of the rectus, one-half inch to the central side of the linea semilunaris, rarely ever going through the transverse muscles or making the McArthur or McBurney incision. When the peritoneum is opened the finger is pressed down into the iliac muscles, drawn along the vessels upward until it hooks or lifts the ileum. This is followed out to the caput coli, the appendix located, freed, its mesentery compressed with heavy clamp forceps, and a catgut ligature placed in the crease. The mesentery is then divided, the base of the appendix is clamped with a heavy hæmostat, and a catgut ligature placed in the crease.

The appendix is then divided close to the ligature and embedded by two rows of fine linen sutures, always beginning on the meso-appendical line of the appendix; each row consists of three or four

continuous stitches; the stump is dropped. This is the procedure in the intermediate operation.

In the operations where infection is present, immediately after opening the abscess a coffer-dam, made of strips of gauze two and a half inches wide, is packed in on all sides of the infected area and retained there until the operation is completed. The abscess is then opened, the pus sponged out, the appendix located, if possible, and the adhesions gently liberated until a ligature can be placed upon the mesentery of the appendix. If the case be one of acute infection and virulent, as interpreted from the symptoms, a ligature is placed on the mesentery and one on the appendix, and both are amputated short, with no effort to embed the stump of the appendix. Notwithstanding the simplicity and this apparent imperfect closure of the appendix, I have not had a single case of fecal fistula requiring an operation for closure. In the septic peritonitis cases the appendix is always removed and a gauze and rubber drainage tube inserted, the latter going into the Douglas cul-de-sac or the vesico-rectal fold. A gauze drain alone in the presence of pus should never be relied on; the same is true of a cigarette drain. Where these are put in for the purpose of transmitting wound suction other than pus to the surface, they fulfil the intention for from eight to twenty hours after their insertion; then they become plugs, not drains. The most reliable and the safest drain is a rubber tube with small perforations. Portions of the coffer-dam around the field of operation may be permitted to remain for days after the operation. This is used less frequently now than it was years ago. The kind of drainage used is governed entirely by the character of the discharge. *All of the acute, severely infected cases are drained, even though the appendix has not perforated.* I consider that the area of the appendix and drainage makes me feel more secure. The drain, under these conditions, may be removed forty-eight to seventy-two hours after the operation. Dry sponges are always used and dry iodoform gauze coffer-dams. Irrigation is never resorted to in any of my abdominal work under any circumstances. The abdomen is closed with the layer sutures. A No. 2 dry catgut is used in the peritoneum, and so placed as to evert the cut edge of the peritoneum out into the abdominal wound and not expose it inside to form adhesions; a No. 4 is used in the aponeurosis of the rectus muscle and horse-hair for the skin. Occasionally a figure-of-8 silkworm-gut is used as an additional support in the aponeurosis of the rectus.

Immediately after the operation the patient is placed in a sitting position, 35 to 45 degrees, and kept there for from two to four days. This is not considered necessary in every case, but it is safer in most.

THE PATHOLOGICAL FINDINGS. It has been my endeavor to treat of the pathological findings in their relations to the symptoms or the stage of the disease, as well as the application of certain operative principles to definite pathological conditions. For a time it was my

practice to describe in detail what I expected to find in opening the abdomen, giving the pathological manifestations in detail. I must frankly confess that I have had many rude awakenings, although the average was fairly accurate. The errors, however, were so significant that had I changed from my rule of prompt action in every case, many lives would have been sacrificed. I was so impressed with these cases that the following expressions were used to the visiting physicians at the operating-table: "We do know from the clinical history, the symptoms, and physical signs that this is a case of appendicitis. We do not know what the tendency of the pathological process in this individual case is, whether it be toward security and repair or toward destruction and danger." A case in point: In a recent consultation, a surgeon of no small pretensions contended that the patient under consideration, then thirty-one hours after the onset of pain, whose pulse in the last six hours had fallen from 110 to 86, and whose temperature had fallen from 102.8° to 100.2°, was progressing favorably and operation might safely be postponed; but on the above rule it was insisted that no one could tell from these symptoms what pathological changes were taking place. The operation was accepted. The appendix was found free from adhesions, a circular gangrene one-half inch in length existed at about its middle, absorption had ceased, the line was clearly defined, and it is unnecessary to mention what would have been the result of delay. An early drop in temperature and pulse and cessation of pain cannot be relied upon as indication of a cessation of danger and must never be interpreted in that way. It only means a cessation of tension and absorption and not a subsidence of pathological changes or their subsequent dangers.

Subsequent Management of Cases. About 80 per cent. of the cases need very little, if any, special attention after the completion of the operation; 10 per cent. demand close watching and 10 per cent. the exercise of the greatest skill to conduct them to a favorable end, and it is to this last 10 per cent. that we will turn our attention. These are the ones that are already severely intoxicated, that have particularly septic processes, that have a local or general septic peritonitis. After the pus tension has been relieved by the opening and drainage and the source of the infection (the appendix) removed, the patient is placed in bed in the sitting (Fowler) position, with a back rest. This favors the settling of the peritoneal and wound exudates into the pelvis to the site of the drainage tube; it is a physiological fact that the pelvic peritoneum absorbs more slowly than the peritoneum above the umbilicus, and, furthermore, that encapsulation is favored by the anatomical structures in the pelvis and the activity of the omentum, sigmoid, and small intestines.

Then attention must be turned to elimination of the products

of infection. This is accomplished, first, by administering large quantities of fluids, rarely intravenous, occasionally subdermally, practically always per rectum and rarely by the stomach. Since we have learned that the stomach does not absorb water, and that in peritoneal inflammations and sepsis it does not readily force the water into the intestine for absorption, we have a good physiological basis for ceasing to give water by the stomach and good physiological explanation for the regurgitation of water administered by the stomach. We know from extensive clinical experience that very great quantities, one pint an hour, may be absorbed by the large intestine. The large quantity of water admitted into the circulation favors osmosis, dilutes the toxins, favors elimination, quiets thirst, lessens nausea, increases peristalsis and expulsion of gases. It is a matter of opinion through which of these channels we derive the greatest benefits. The stubborn fact remains, however, that the patient's comfort is greater, the condition improves, and they get well. Strychnine may have some value as supplementing the proctoclysis. If alcoholics are given at all they should be given by mouth, as they are rapidly absorbed by the stomach. Nuclein and unguentum Credé are beneficial in special cases, the former when the local resistance of the tissues seems to be meagre, the latter in the streptococcic types of infection with its exhilarative nerve phenomena. Calomel should be administered in small and repeated doses until effectual.

IMMEDIATE RESULTS. The immediate results, if taken by hundreds, have varied very materially. The greatest mortality was in the first 100 cases, when it reached 11, and I might also add that in that 100 cases practically every one was an acute infection with pus perforation, more or less peritonitis, etc.; they embraced the cases from March 2, 1889, to July 23, 1893.

The number of deaths in each 100 then gradually decreased until after the meeting of the American Medical Association at Atlanta in May, 1896. It will be recalled that at that meeting there was a great cry against peritoneal operations and particularly against the frequent operations for appendicitis. This was heralded through the journals, and the general practitioner, believing it represented the consensus of opinion of the majority of surgeons, who were in close contact with the public, or general practitioners, accepted it as a fact and hesitated before advising operations when the unquestioned symptoms of appendicitis were present. The price in lives was great. The arguments urged represented the ideas of men who conducted large clinics, and not in close contact with the people or with the every-day practitioner, who saw mostly intermediate cases and did not realize the virulence, rapidity of destruction and danger, as well as the consequent vital importance of prompt and early action in the acute infective stage. In the year following this meeting the mortality reached 7 in 100. Then

there was another gradual decline, as the error of the position was recognized, until a second epidemic of so-called conservatism ("expectant treatment") passed over the country and carried with it its quota of deaths, when the mortality reached 6 in 100. For the last four years there has been a steady decline in the number until in the last 100 operations there was a mortality of 2 per cent. One of these deaths was due to an ileus of the small intestine high up, bearing no relation either in adhesions or otherwise to the appendical operation, and the other a death from a double pneumonia six days after the operation for general septic peritonitis of appendical origin, the peritonitis having entirely subsided.

It seems to me that every death from appendicitis is chargeable directly to the people, either for not calling in the physician sufficiently early after the onset of symptoms, or to the physician and surgeon for not acting promptly when they are called. I am sorry to admit that the latter represents the greater percentage. We should have no deaths from appendicitis, but *we are having them.* We should accept the force of numbers and experience to guide us against the culpable if not criminal error of delay in this class of cases. It is not necessary that every physician should lose a case so he may learn the lesson, any more than a doctor should lose a case under chloroform anæsthesia before he learns either to abandon it or have it administered by competent hands.

FINAL RESULTS. The final results in appendicitis, as a whole, are very gratifying. When a patient has had the appendix removed, the rule is that he recovers from the operation, has as good if not better health than he had before, and is free from pain and inconvenience in the right hypochondriac region or disturbance of his digestive tract as a result of the disease or the operation. It is surprising how completely extensive adhesions, the result of diffuse and extensive inflammatory processes, disappear soon after the focus of infection is removed and the source of irritation eradicated. Prolonged packing and drainage tend to the formation of more firm adhesions than usually result from the inflammatory processes with their adhesions in limiting the infective process. With the exception of the adhesions following tuberculosis in the peritoneum, there is no other lesion in which the adhesions so rapidly and completely disappear as those that exist around an inflamed appendix. If the appendix has perforated and a secondary perforation has taken place into the intestine, a firm and organic adhesion of the appendix may remain, uniting it to the intestine, the caput coli, or the ileum, as the case may be. It seems to me even these rapidly absorb, so that in an abdomen where six weeks previous an operation had been performed for the drainage of a large periappendical abscess, with extensive suppuration and adhesions, almost every evidence of the disease, except a fluffy, velvety condition of the peritoneum, had disappeared. This is an every-day observation.

The regenerative power of the epithelium over the intestine is vast in the average case. Occasionally we find a tendency to keloid formations in the peritoneum, or to the formation of cord-like adhesions which develop into strong, firm bands of sufficient power to lift the patient from the table. Fortunately these are rare. If the appendix remains and becomes a chronic source of irritation after an attack of appendicitis, the adhesions often continue and remain there as a protection against a subsequent attack, rendering the danger in a subsequent attack less in these cases. If we could only tell from the symptoms that they existed, what a comfort it would be in recurrences!

HERNIA. Much has been written on the subject of hernia following the operation for appendicitis, and many suggestions have been made concerning the original incision. It seems that one of two incisions should be adopted: either the gridiron incision of McBurney and McArthur, or the incision through the rectus muscle. The gridiron incision has the advantage of retaining the muscle fibres without injury while it divides the muscular sheath, which is often strong, at the point of separation of the fibres. It has the disadvantage of confining the operator to a comparatively small field after the abdomen is opened, unless he is willing to sacrifice the muscle by division. This is not such a serious matter as we formerly believed. Muscle fibres may be divided in a direct transverse line if they are again brought into apposition and the full strength of the muscle retained. This is seen in division of the biceps, triceps, and the abdominal rectus. It leaves a depression and a deformity, but no deficiency in support. The other incision is one to the central side of the linea semilunaris, going through the rectus muscle. It has the advantage of permitting the incision to be elongated from the symphysis to the costal arch without the division of muscle fibres. It has the further advantage of permitting the peritoneal and the muscular aponeurosis to be sutured without including the muscle fibres and, at the same time, produces approximation of the slit muscular fibres side to side in their long axis, the sutures being applied in layers. The accurate apposition of the aponeurosis of the rectus with a catgut or figure-of-8 suture practically guarantees against a hernia. In the class of cases in which drainage is necessary there is a defect in the approximation of the aponeurosis of the rectus or of the muscle fibres if the gridiron incision be made, which leaves a weakness in the wall; this, in a considerable percentage of cases, results in hernia. Prolonged drainage is not so frequently necessary as was formerly supposed. A figure-of-8 suture, to be tied later, can be used for the approximation of the cut edges of the aponeurosis at this point. It should be allowed to remain loose until the drainage is removed, and then it may be drawn taut and good apposition secured, which will lessen the likelihood of hernia, though it will not prevent an

occasional one. If the postoperative hernia is tending to increase, it should be closed with an edge-to-edge union of the aponeurosis, or a flap operation of the aponeurosis, after the method of Mayo for umbilical hernia. Hernia bears close relation to drainage in all locations of incision in the abdominal wall.

OBLIGATIONS OF THE GENERAL PRACTITIONER. My early experience with appendicitis occurred when I was doing general practice, the first operation having been performed at Cook County Hospital on March 2, 1889, which I believe was the first one in this country under the new idea of the surgical treatment of appendical and perityphlitic affections or induration—*i. e.*, operating in the absence of phlegmon, but on the theory that the primary lesion was an infection and suppuration in the appendix and the perityphlitis, a secondary extension of this infection. Periappendical abscesses with phlegmon and induration had been drained and earlier intervention urged in most forceful language by Bull and others preceding that time. (The report of this operation was made to the Chicago Medical Society, November 4, 1889.) This operation was performed after reading the able paper of Krufft, which I think did more to stimulate early operations on the appendix at that time than any other paper that appeared. Therefore, I make no apology for speaking of the obligations of the general practitioner in appendicitis. In a large number of the early cases of appendicitis I was the first doctor called, and after a few experiences, which were so forceful in their instruction, it required no courage on my part to insist on the operation. Many of the first hundred operations were performed on the kitchen-table, with the ordinary aseptic conditions that could be secured in a twenty-minute or half-hour preparation. In a large number of them there was no trained nurse, no cumbersome excessive surgical paraphernalia, avoiding the loss of time which these entailed, hastening the liberation of the pus or the removal of the appendix, as the case might be. The facts gleaned at that time made me realize, as no subsequent experience has, the degree of destruction that can occur in a very limited number of hours with virulent infection of the appendix. Indeed, I was frequently asked, "Where do you get all of the appendicitis cases?" with a look and an inflection that did not indicate the utmost confidence in my diagnostic ability or the integrity of the numerals; but that is ancient history. Appendicitis has come and it has come to stay, and the responsibility of its immediate recognition is on the general practitioner. It seems to me that a man has no more right to hope that an inflammation of the appendix, once instituted, will tend to a favorable termination than he has to hope that an acute, infective osteomyelitis of the femur, or a phlegmon of the thigh, will recover without liberation of the pus.

Can the general practitioner make an early and accurate diagnosis? Yes. This is admitted by all at the present time. Is he

justified in hoping that the case will subside and go through to a favorable termination? No! We will admit that 80 per cent. of the cases recover from the attack; that 20 per cent., approximately, of the cases either die in the first attack or have recurrences. If he decides to wait, it is with the understanding that he is willing to sacrifice that percentage of his patients. All aid can as readily be timely as untimely. It seems to me that as a general practitioner I would feel personally responsible for the death of a patient suffering from a typical attack of acute appendicitis, if I had the case from the beginning and was called early—that is, in the primary pain. I might endeavor to ease my conscience by urging certain reasons for withholding action, or by stating that I was following Smith's or Brown's or Jones' plan of treatment, but the ever-living witness of inner judgment would be crying out against me. There is no plan of treatment which controls the pathological processes. There is no plan of medical treatment which materially lessens the risk. There is no system of beating the mortality percentages in appendicitis.

I wish to express my high appreciation of the strong position taken by many of the ablest medical men in insisting that they have no power of control over the disease, and that it is essentially from its initiation a surgical condition. Indeed, these men outrank many of the surgeons in their judgment and in their appreciation of the urgency of immediate action.

OBLIGATIONS OF THE SURGEON. The obligation of the surgeon is to conduct a case of appendicitis to a favorable termination *with the least hazard or risk to the patient*, regardless of his personal or professional feeling or of the praise or condemnation of his conduct. We all meet cases where we would readily shirk the responsibility of action, but we have no license in the role of surgeons if we take that position. We all meet cases in which we would prefer to have one clean operation and be through with the case, but that must never be done where it involves additional hazard to the patient. A second operation with a living patient at the end is better than a primary radical operation with a mortality of two or three in a hundred as the price. This involves the element of individual judgment and cannot be adequately described. The man who is having more than three or four deaths in a hundred operations for appendicitis is either receiving his patronage from incompetent and procrastinating medical men, or he is doing too much manipulating in the peritoneal cavity under unfavorable pathological conditions. There are practitioners who recognize the earliest symptoms, and, indeed, I recall one who forced me on two occasions to operate where I was not satisfied of the diagnosis, and on each occasion he was correct.

The transportation of patients after the onset of an attack of appendicitis is an important factor, and one that comes under the

class of surgeon's obligations. If the case is brought to my attention before the thirtieth hour I feel that the patient can be transported to the hospital with safety. If, on the other hand, it is after that time it is safer to have the operation performed in the environment of his home, whatever that home may mean. If the patients are to be transported, they should be kept in the sitting position on the stretcher. Once the surgeon has operated he is obligated for all of the details of the case to a complete convalescence. If the associate is sufficiently competent to care for the patient under his instruction, well and good; otherwise he must see to it himself that all of the details are carried out to a complete and perfect convalescence.

PSEUDOMELANOSIS OF THE HÆMOLYMPH GLANDS.

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The writer's contention that lymphoid structures containing sinuses filled with red blood cells and showing evidences of extensive phagocytic hæmolysis are constantly present in the human retroperitoneal tissues has been widely accepted during the last two years. His statement that the hæmolytic activity of these organs is relatively greater than that of the spleen has been recently confirmed by Lewis,[1] who made comparative counts of the number of phagocytes containing red cells and pigment occurring in equal fields of the spleen and hæmolymph nodes. A number of organs were cut and several sections from each examined. In each section as many isolated fields as possible were taken with a $\frac{1}{6}$ objective, and the number of phagocytes containing red cells or pigment particles in each field were counted. Estimates were also made of the number of red cells enclosed within the phagocytes. From the results of these observations a few examples may be taken: In the case of the rat the proportion of phagocytes in the spleen was 1.6 to 93 approximately in the hæmolymph nodes; in the rabbit 1.2 in the spleen to 60 in the hæmolymph nodes; in the sheep 0.5 in the spleen to 31 in the hæmolymph nodes. In all cases the hæmolymph nodes showed by far the greatest amount of phagocytosis per average field.

Similar results have been obtained by the writer, and the suggestion made by him in 1902 that these nodes might properly be styled "hæmolytic glands" acquires additional force. Further confirmation of the marked hæmolytic activity occurring in these glands is shown by the striking and very interesting findings in the

[1] Journal of Anatomy and Physiology, vol. xxxviii,